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(CASE REPORT)

Maxillary Ameloblastoma: A Case Report of a Rare Tumor and a Brief Review of the Literature

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Abstract

Ameloblastoma is a benign yet locally aggressive odontogenic tumor, with the maxillary presentation being a rare and clinically challenging entity due to its proximity to vital midfacial structures and tendency for silent, rapid expansion. This report detailed the case of a 23-year-old female who presented with an eight-month history of progressive left-sided facial swelling and subsequent infraorbital nerve paresthesia, initially misdiagnosed as a dental issue. Key diagnostic findings included a large, expansile, multilocular radiolucent lesion on panoramic and CT imaging, exhibiting the classic "soap bubble" appearance. Histopathological examination confirmed the diagnosis of conventional (multicyclic) ameloblastoma, follicular subtype, characterized by islands of odontogenic epithelium with peripheral palisading and reverse polarity.

The management involved a radical infrastructure maxillectomy with histologically confirmed negative margins, followed by immediate microvascular reconstruction using a vascularized fibular free flap. The patient achieved an excellent functional and aesthetic outcome, with no evidence of local recurrence at the three-year follow-up. This case underscores the critical importance of maintaining a high index of suspicion for persistent midfacial swellings. It validates the use of radical surgical resection combined with immediate microvascular reconstruction as the gold standard for achieving oncological safety and optimal quality of life in young patients with extensive maxillary ameloblastoma.

Keywords: Ameloblastoma; Benign; Aggressive; Odontogenic Tumor; Maxilla

1. Introduction

Ameloblastoma is recognized as a benign but locally aggressive odontogenic neoplasm that primarily affects the mandible, with the maxilla being involved in only approximately 15% of cases. [1] Despite its benign histological classification, the maxillary variant poses a more formidable clinical challenge due to the thin maxillary cortical plates and its proximity to the paranasal sinuses, orbit, and skull base. [2] These anatomical factors facilitate the silent, rapid spread of the tumor, often leading to late-stage presentations and a higher risk of recurrence compared to its mandibular counterpart. [3] Although ameloblastoma is typically a benign but locally aggressive tumor, a study of 1,626 cases from East China by Liu W et al. reported that it presented as a malignant tumor in 3.4% of cases, and a recurrence rate of 17.2%. [19]

Historically, the management of maxillary ameloblastoma is complicated by the delicate balance between achieving radical surgical clearance and preserving essential midfacial functions and aesthetics. [4] A significant knowledge gap

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persisted regarding the optimal surgical margins and the long-term efficacy of immediate microvascular reconstruction in young patients, where the impact on quality of life is most profound. [5] We report this case to highlight the successful management of a large maxillary ameloblastoma through an infra- and supramaxillary maxillectomy and immediate fibular free flap reconstruction, emphasizing the importance of a multidisciplinary approach in addressing this rare and aggressive entity.

2. Case Presentation

2.1. Clinical presentation

A 23-year-old female noticed progressive left-sided facial swelling over 8 months. She first attributed the subtle swelling to dental issues. She sought care from her general dentist, who performed a routine extraction of her upper left second molar for presumed periapical pathology. However, the swelling continued to enlarge despite the extraction. Over the subsequent three months, she developed increasing facial deformity with visible left maxillary prominence, mild nasal obstruction on the ipsilateral side, and paresthesia of her left upper lip and cheek. She also reported loosening of adjacent teeth and occasional epistaxis. The patient delayed seeking further medical attention due to financial constraints and fear of a potential diagnosis. She finally presented to the oral and maxillofacial surgery clinic when the facial asymmetry became cosmetically distressing, and she developed difficulty with mastication and speech articulation.

2.2. History, physical examination, and laboratory studies

The patient had no significant past medical history and was not taking any medications. She denied tobacco use, consumed alcohol socially, and had no history of radiation exposure. Her family history was unremarkable for jaw tumors, genetic syndromes, or malignancies. She had no personal history of previous odontogenic lesions or multiple dental anomalies. Her menstrual history was regular, and she was nulliparous.

On physical examination, she appeared well-nourished and in no acute distress. Vital signs were within normal limits. Facial examination revealed marked asymmetry with a 5×4 cm firm, non-tender swelling of the left maxilla extending from the midface to the zygomatic region. The overlying skin was intact without erythema, warmth, or visible lesions. There was mild obliteration of the left nasolabial fold. Palpation revealed a bony-hard, non-fluctuant mass with smooth borders and no crepitus. Intraoral examination showed expansion of the left maxillary alveolar ridge with intact but thinned overlying mucosa. The mass extended from the canine region posteriorly to the tuberosity. The left upper first premolar and first molar exhibited grade II mobility. There was no mucosal ulceration, bleeding, or necrosis. The palate showed buccal cortical expansion with some palatal displacement. Sensation testing revealed hypoesthesia in the distribution of the left infraorbital nerve.

Laboratory studies, including complete blood count, comprehensive metabolic panel, and coagulation profile, were within normal limits, indicating no systemic abnormalities or metabolic disturbances.

2.3. Radiologic studies

Panoramic radiography demonstrated a large, expansile, multilocular radiolucent lesion involving the left maxilla, extending from the lateral incisor region to the maxillary tuberosity. The lesion measured approximately 5×4 cm with well-defined scalloped borders and internal septations creating a "soap bubble" appearance. There was significant cortical thinning with maintained cortical integrity, root resorption of the first premolar and first molar, and displacement of the maxillary sinus floor superiorly. Computed tomography (CT) with contrast revealed a heterogeneous hypodense lesion with extensive expansion and remodeling of the maxilla. There was a perforation of the buccal cortex in several areas, but preservation of the lingual cortex. The mass extended into the inferior portion of the maxillary sinus without significant sinus opacification. No pathological lymphadenopathy was identified. Post-contrast images showed minimal peripheral enhancement, with no significant internal vascularity.

Magnetic resonance imaging (MRI) showed a predominantly T1 hypointense and T2 hyperintense multiloculated mass with thin enhancing septations. The solid components demonstrated intermediate signal intensity on both sequences. There was no intracranial extension, orbital involvement, or infiltration of the pterygoid plates. The lesion maintained a well-defined interface with adjacent structures despite its size. Radiological differential diagnosis included ameloblastoma (favored given the classic multilocular appearance and patient age), odontogenic keratocyte, central giant cell granuloma, odontogenic myxoma, myeloblastic fibroma, and myeloblastic carcinoma. The extensive cortical expansion, multilocular pattern, and patient demographics strongly suggested ameloblastoma, though histological confirmation was required for a definite diagnosis due to the rarity of this tumor and to rule out myeloblastic carcinoma.

2.4. Tumor board discussion

The multidisciplinary tumor board convened with oral and maxillofacial surgeons, head and neck oncologists, radiation oncologists, pathologists, and reconstructive surgeons. Discussion centered on surgical approach, extent of resection, and immediate versus delayed reconstruction. The board emphasized that, despite their benign histology, Amel blastomas exhibit locally aggressive behavior and high recurrence rates following conservative treatment. Given the tumor size, cortical perforation, and the patient's young age with long life expectancy, the consensus favored radical surgical excision over conservative enucleation or curettage. The team debated between a partial maxillectomy with 1-2 cm margins and an infrastructural maxillectomy.

Concerns regarding facial aesthetics, functional outcomes, and quality of life in this young female patient were thoroughly discussed. The decision was made to proceed with an infrastructure maxillectomy with immediate microvascular fibular free flap reconstruction and dental rehabilitation planning. Radiation therapy was not recommended, given the benign nature and radiation resistance of Amel blastomas. Close long-term surveillance was deemed essential.

2.5. Tissue diagnosis

An incisional biopsy was performed prior to definitive surgery. Gross pathology of the biopsy specimen showed firm, tan-white tissue fragments with a rubbery consistency and small cystic spaces containing straw-colored fluid. Microscopic examination revealed classic features of follicular ameloblastoma. The tumor consisted of islands and nests of odontogenic epithelium within a fibrous connective tissue stroma. The peripheral cells of these islands showed columnar and cuboidal morphology with hyperchromatic nuclei exhibiting reverse polarity (nuclei polarized away from the basement membrane), resembling ameloblasts. The central portions of the epithelial islands contained loosely arranged stellate reticulum-like cells. Multiple microcysts were present, some containing eosinophilic fluid. The stroma showed minimal inflammatory infiltrate. Importantly, there was no cytological atypia, increased mitotic activity, perineural invasion, or stromal invasion that would suggest myeloblastic carcinoma. The epithelial-stromal interface was well-defined without destructive infiltration. (Figure 1 A, B, C)

Histological differential diagnosis considered ameloblastoma (to be confirmed with immunohistochemistry), myeloblastic carcinoma (excluded due to absence of malignant features), adenomatoid odontogenic tumor (excluded by lack of duct-like structures and calcifications), and myeloblastic fibroma (excluded due to absence of ectomesenchyme component and patient age). Immunohistochemistry (IHC) demonstrated: CK19 positive in epithelial components, p63 positive in peripheral columnar cells, SOX2 positive in stellate reticulum areas, and a Ki-67 proliferation index of approximately 3-5% (low). These markers supported the lesion's benign nature.

Molecular studies were not performed as the diagnosis was established. The final diagnosis was conventional (multicyclic) ameloblastoma, follicular subtype, consistent with a benign locally aggressive odontogenic neoplasm.

2.6. Treatment, outcome, follow-up, and three-year status

The patient underwent left infraorbital maxillectomy under general anesthesia with nasotracheal intubation. The surgical team achieved en bloc resection with histologically confirmed negative margins (minimum 1.5 cm). Immediate reconstruction was performed using a vascularized fibular free flap with microvascular anastomosis to the facial vessels. The fibula was contoured and osteotomized to recreate maxillary architecture. Osseointegrated dental implants were placed in a delayed secondary procedure six months postoperatively. The immediate postoperative course was uncomplicated. The patient received prophylactic antibiotics and anticoagulation. The flap remained viable with good Doppler signals throughout the monitoring period. She was discharged on postoperative day 7 with instructions for a soft diet, oral hygiene protocols, and wound care. The follow-up protocol included clinical examination and imaging every 3 months for the first 2 years, then every 6 months thereafter. Surveillance imaging alternated between panoramic radiography and CT scans to monitor for recurrence.

Three-year status: The patient demonstrated excellent functional and aesthetic outcomes. The reconstructed maxilla showed complete bony consolidation with well-integrated implants supporting a fixed prosthetic rehabilitation. She regained normal masticatory function and speech. Facial symmetry was well-maintained with acceptable cosmetic results. Serial imaging showed no evidence of local recurrence. Hypoesthesia of the infraorbital nerve gradually improved to approximately 70% of normal sensation. She returned to full professional and social activities with high satisfaction scores on quality-of-life assessments. She remained under continued annual surveillance given the potential for late recurrence.

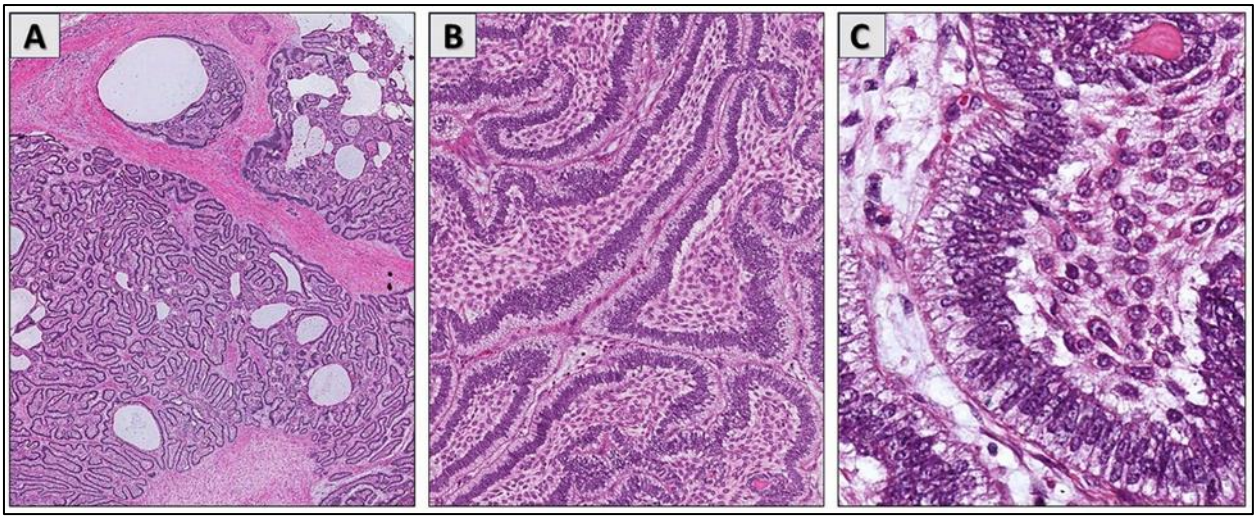

Figure 1 Microscopic examination of the excised ameloblastoma

- 1A: Low power view showing islands and nests of odontogenic epithelium within a fibrous connective tissue stroma (H&E X20).
- 1B: Intermediate power view showing well-defined epithelial-stromal interface without destructive infiltration (H&E X40).
- 1C: High power view showing peripheral columnar cells with hyperchromatic nuclei exhibiting reverse polarity (nuclei polarized away from the basement membrane with perinuclear clearing), resembling ameloblasts. (H&E X60)

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification)

The approach to the term “ameloblastoma” has changed significantly over nearly one and a half centuries. The first suggested name of this tumor was adamantinoma, a term coined by Malassez in 1885. [6] However, the term was later excluded on the basis that it implies mainly the formation of hard tissue, an improbable occurrence in ameloblastoma.

The classic ameloblastoma is another significant nomenclature change. In WHO 2017, 4th ed., the term was revised from "Solid or multicystic ameloblastoma" to "Conventional ameloblastoma." [7] This change was made to avoid confusing this entity with unicystic ameloblastoma. In this context, conventional ameloblastoma with macrocystic change has previously been termed cystic ameloblastoma in various publications. [7] However, this term was also abandoned because it could lead to confusion with unicystic ameloblastoma. Additionally, desmoplastic ameloblastoma has been reclassified. This entity is no longer considered a blueprint member; it was previously recognized as a separate and distinct subtype of ameloblastoma in the WHO 2005 3rd edition. [8] However, it is no longer recognized as a separate entity and is now regarded as a microscopic aspect of conventional ameloblastoma in the WHO 2017 4th edition. The most useful reclassification may be that for metastasizing ameloblastoma. This entity has previously been designated as metastasizing (malignant) ameloblastoma and placed among the malignant odontogenic tumors in the WHO 2005, 3rd Edition. [9] Nevertheless, this tumor was delisted from malignant odontogenic tumors and listed under benign odontogenic tumors in WHO 2017 (4th Edition). [8]

The World Health Organization (WHO) classification of odontogenic tumors, most recently updated in 2022, categorizes ameloblastoma into several distinct types: conventional, unicystic, extraosseous/peripheral, and the newly recognized adenoid ameloblastoma. Conventional ameloblastoma, as seen in our patient, is characterized by its multicystic or solid growth pattern and is notorious for its locally invasive behavior. [8] Ameloblastic carcinoma is a rare primary odontogenic carcinoma of the jaw that exhibits ameloblastic differentiation, cytologic atypia, increased mitotic figures, expansion of the basal layer, and often punctate tumor necrosis. [1] Understanding these foundational aspects is essential for clinicians to maintain a high index of suspicion when encountering persistent, painless midfacial swellings.

Epidemiologically, the tumor demonstrates a wide age distribution, typically peaking in the third to fifth decades of life, with no significant gender predilection. [10] While the mandible is the site of origin in approximately 80-85% of cases,

the maxilla remains a rare but clinically significant location, often involving the posterior regions. [11] Risk factors remain largely elusive. However, some studies suggested a potential link to dental trauma or chronic irritation, yet most cases appeared to arise sporadically from the epithelial remnants of the dental lamina. [10]

3.2. Pathogenesis, Pathophysiology

The pathogenesis of ameloblastoma is rooted in the neoplastic transformation of odontogenic epithelium, specifically originating from the remnants of the dental lamina (rests of Serres), the enamel organ, or the epithelial lining of odontogenic cysts. [1] At the molecular level, recent advances have identified distinct mutational profiles that dictate tumor behavior and location. While BRAF V600E mutations predominated in mandibular lesions, maxillary ameloblastomas frequently harbor mutations in the SMO (Smoothened) gene, a key component of the Hedgehog signaling pathway. [12] [13] These genetic alterations drive uncontrolled cellular proliferation and survival. Pathophysiologically, the tumor's locally aggressive nature is facilitated by its ability to induce significant bone resorption. Ameloblastoma cells are found to express high levels of Receptor Activator of Nuclear Factor Kappa-B Ligand (RANKL) and Matrix Metalloproteinases (MMPs), particularly MMP-9. [14] This molecular signaling stimulates osteoclastogenesis and extracellular matrix degradation, allowing the tumor to infiltrate the surrounding trabecular bone and, in the case of the maxilla, to easily penetrate the thin cortical boundaries to invade adjacent anatomical spaces. [12]

3.3. Comparative analysis of our case with the existing literature. (Clinical-radiology-pathology, lab, diagnosis-management, and outcome)

The clinical presentation of our 23-year-old patient, characterized by a slow-growing, painless maxillary swelling initially misdiagnosed as a dental issue, mirrored the classic literature narrative. Maxillary ameloblastomas are frequently reported to be "silent" in their early stages, often leading to significant delay in diagnosis. In a retrospective study of 13 cases by Nastri et al., the lack of early symptoms was highlighted as a primary reason for the advanced size of these tumors at presentation. [15] Our patient's eight-month delay and subsequent presentation with facial asymmetry and infraorbital hypoesthesia are consistent with the aggressive local expansion noted in other series, such as that by Evangelou et al., which emphasized that maxillary lesions often erode the thin cortical plates and invade the paranasal sinuses more readily than mandibular lesions. [2]

Radiologically, the "soap bubble" multilocular appearance observed in our patient’s panoramic and CT imaging is a hallmark of conventional ameloblastoma. [15] While unicystic variants are more common in younger populations, the multicystic pattern seen here is typical for the follicular subtype. [16] The use of MRI in our case provided critical information regarding the soft-tissue interface and the absence of orbital or skull-base involvement, a diagnostic step strongly advocated by Minami et al. for large maxillary tumors to delineate the extent of resection. Our findings of T2 hyperintensity and peripheral enhancement aligned with the imaging characteristics described in their series of 26 patients. [17]

The management of this case represents a shift toward radical primary surgery, which was increasingly supported by evidence-based algorithms. [3] Historically, conservative treatments such as enucleation and curettage were associated with recurrence rates of 60-90% in the maxilla. [5] In contrast, radical resection with 1.5-2 cm margins, as performed in our patient, was shown by Carlson and Marx to significantly reduce recurrence to less than 15%. [18] The decision for an infrastructure maxillectomy followed by immediate microvascular fibular free flap reconstruction was particularly noteworthy. While some authors previously favored delayed reconstruction to monitor for recurrence, recent series have demonstrated that immediate reconstruction in young patients does not compromise surveillance and vastly improves functional and psychosocial outcomes. [5] Our patient’s three-year recurrence-free status and excellent aesthetic result validate this aggressive yet reconstructive-focused approach, which remains the gold standard for large, locally invasive maxillary ameloblastomas. [3] [5] [18]

4. What Have We Learned from This Case?

This case provided several critical clinical insights into the management of maxillary ameloblastoma. First, it underscores the need to maintain a high index of suspicion for persistent midfacial swelling, as early symptoms are often subtle and easily attributed to common dental pathologies. The delay in diagnosis in this young patient highlights the need for clinicians to perform thorough radiological evaluations, including CT and MRI, when routine dental treatments fail to resolve clinical symptoms.

Second, the successful outcome reinforced the change in thinking toward radical primary surgical resection for maxillary lesions. Given the anatomical complexity and the aggressive nature of the tumor in this location, conservative approaches are deemed insufficient and carry an unacceptably high risk of recurrence.

Third, the case demonstrated that immediate microvascular reconstruction using a fibular free flap was not only feasible but highly beneficial in young patients. It allowed restoration of facial symmetry, masticatory function, and speech, which were vital to the patient's quality of life and psychological well-being.

Finally, the importance of a multidisciplinary tumor board was evident, as it ensured a comprehensive treatment plan that balanced oncological safety with functional rehabilitation. Long-term, vigilant surveillance remained mandatory, as late recurrences are a known characteristic of this enigmatic tumor.

5. Conclusion

This case of a large maxillary follicular ameloblastoma in a 23-year-old female served as a strong reminder of the tumor's locally aggressive behavior and the diagnostic challenges it posed in the midfacial region. We presented this case to emphasize that, despite its benign histological classification, the maxillary variant required a radical surgical approach to ensure long-term disease-free survival. The successful integration of an infrastructure maxillectomy with immediate microvascular fibular free flap reconstruction demonstrated that oncological safety and functional restoration could be achieved simultaneously, even in extensive lesions. This report adds to the growing body of evidence supporting immediate reconstruction as a viable and superior option for maintaining the quality of life in young patients. By documenting this successful multidisciplinary management and the three-year recurrence-free outcome, we aimed to provide clinicians with a practical framework for addressing similar rare and challenging odontogenic tumors, ultimately benefiting the broader medical community through shared clinical experience and improved patient care standards.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified prior to analysis. No patient contact or intervention was involved, and informed consent was not required in accordance with institutional policies.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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